Ruptured Giant Basilar Artery Aneurysm in a Comatose Adolescent: Successful Obliteration Using Intraoperative SSEP, BAER, and MEP Monitoring

A Case Report

C.I. NWAGWU, M.S. MATHEWS, J.A. SCOTT*, A.J. DENARDO*, T.G. HORNER*

Department of Neurosurgery, University of California (Irvine), Orange, California; Indianapolis Neurosurgical Group, Indianapolis, Indiana.*

Key words: giant basilar aneurysm, aneurysm coiling, SSEP, BAER, MEP

Summary

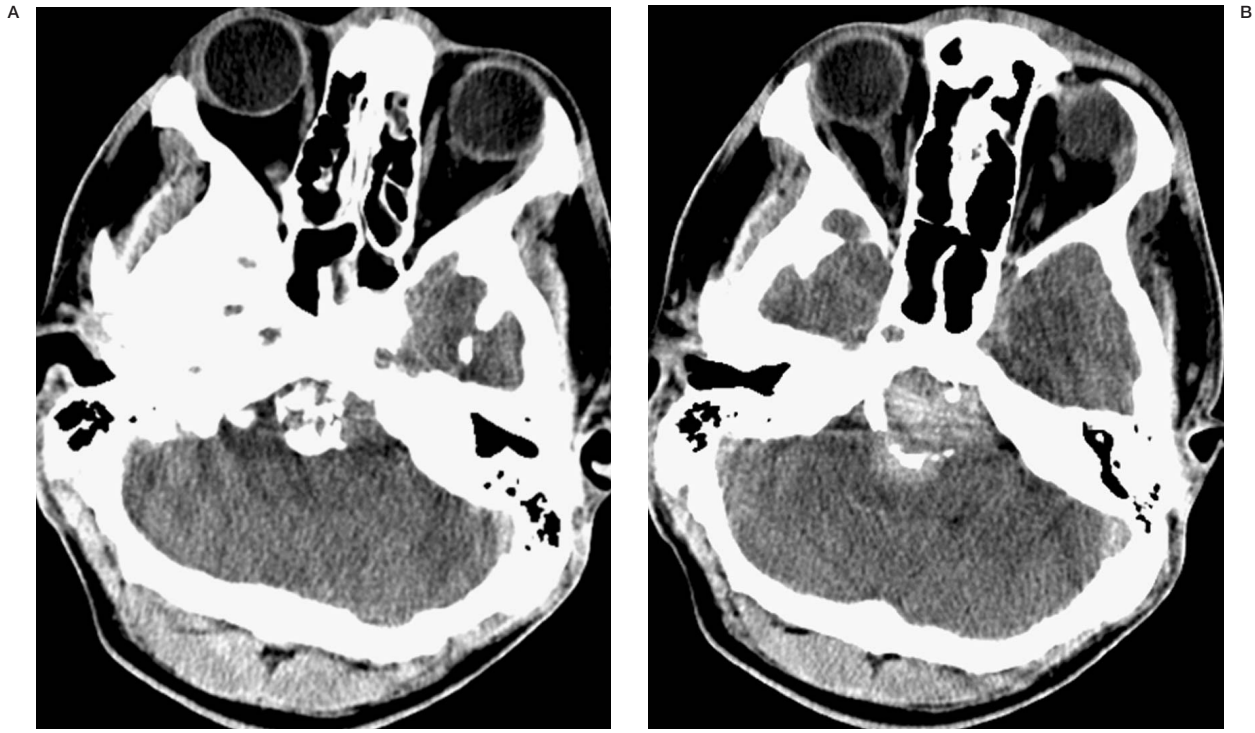
Giant basilar aneurysms are infrequently seen in children. We present the endovascular management of an adolescent who presented comatose with pinpoint pupils due to a ruptured giant basilar trunk aneurysm. A noncontrast head CT disclosed a large prepontine lesion with brainstem hemorrhage. Catheter angiography showed a 4.5 cm irregular, fusiform basilar trunk aneurysm. With SSEP, BAER, and MEP monitoring, the patient underwent bilateral temporary vertebral artery occlusion, followed by GDC embolization of the aneurysm. Postprocedure internal carotid angiograms showed adequate blood supply to the basilar apex via patent posterior communicating arteries. On postprocedure day two, the patient was following commands.

The remainder of his hospital course was uneventful. Postoperative angiograms showed no residual filling of the aneurysm. At 12 months the patient was neurologically intact and at baseline function as an honor student and follow-up angiogram showed persistent occlusion of the aneurysm from the circulation. Successful endovascular treatment has been considered a less invasive and safer alternative to surgical management of some complex vascular lesions. While most reports on reversing basilar artery flow have been carried out in awake patients with neurological examinations, this is not possible in a patient presenting in a comatose state. This report suggests that SSEPs, BAERs and MEP may be of use in such patients in safely carrying out basilar artery occlusion.

Introduction

Basilar trunk aneurysms are rare lesions and account for less than 1% of intracranial aneurysms. Approximately 8% of giant aneurysms occur in the posterior circulation [1]. Among vertebrobasilar aneurysms approximately 8% are on the basilar trunk [7]. Common presenting symptoms of basilar trunk aneurysms are related to brainstem compression from the mass effect of an enlarging aneurysm, intraluminal thrombosis and cerebral infarction or from rupture with SAH as an acute presentation.

Clinical features can vary from being entirely asymptomatic to deep coma. Such aneurysms are rare in children. Surgical clipping of basilar trunk aneurysms is technically challenging and carries significant risks due to difficult exposure and vulnerability of the vital neighboring structures. Endovascular occlusion has emerged as an effective alternative to surgery in treating fusiform basilar aneurysms. Several studies have reported successful obliteration of giant posterior circulation aneurysms using endovascular coiling, vertebral occlusions, and



flow reversal. We report the case of an adolescent presenting in deep coma and mixed posturing secondary to rupture of a 4.5 cm giant fusiform basilar aneurysm.

Case Report

A 16-year-old, right-handed male with no significant medical history was found unresponsive on his bedroom floor by his mother after she heard him fall. He was immediately brought into the emergency department where he was found to have pinpoint pupils, ocular bobbing, and mixed posturing, with no eye opening. A noncontrast head CT scan disclosed a large prepontine lesion with thrombus, and acute hemorrhage in the periphery (figure 1). Catheter angiography showed a 4.5 cm irregular, fusiform, partially thrombosed basilar trunk aneurysm that extended along a substantial length of the distal basilar artery (figure 2). Considering the fusiform and complex nature of the aneurysm, as well as the anticipated technical difficulty of surgical trapping, we decided to treat the aneurysm through an endovascular approach.

With SSEP, BAER, and MEP monitoring, the patient underwent the following: using 6-French guide catheters (Envoy, Cordis, Miami Lakes, FL, USA) both vertebral arteries were selected. Hyperform balloons (4 x 7 mm) were then advanced to the distal vertebral artery (proximal to the basilar origin and distal to the PICA origin), and both vertebral arteries sequentially occluded. No significant changes in neurophysiologic parameters were observed during this test occlusion. Angiography through both internal carotid arteries during vertebral artery occlusion demonstrated preserved flow to the posterior cerebral arteries and distal basilar artery through posterior communicating arteries (figure 3).

The aneurysm was then embolized using detachable platinum microcoils with preservation of both vertebral arteries and the basilar origin. Postprocedure internal carotid angiograms showed adequate perfusion to the basilar apex with successful obliteration of the aneurysm (figure 4).

On postprocedure day two, the patient was following commands. The remainder of his hospital course was uneventful. Postoperative angiograms showed no residual filling of the aneurysm. At 12 months the patient was neurologically intact and at baseline function as an

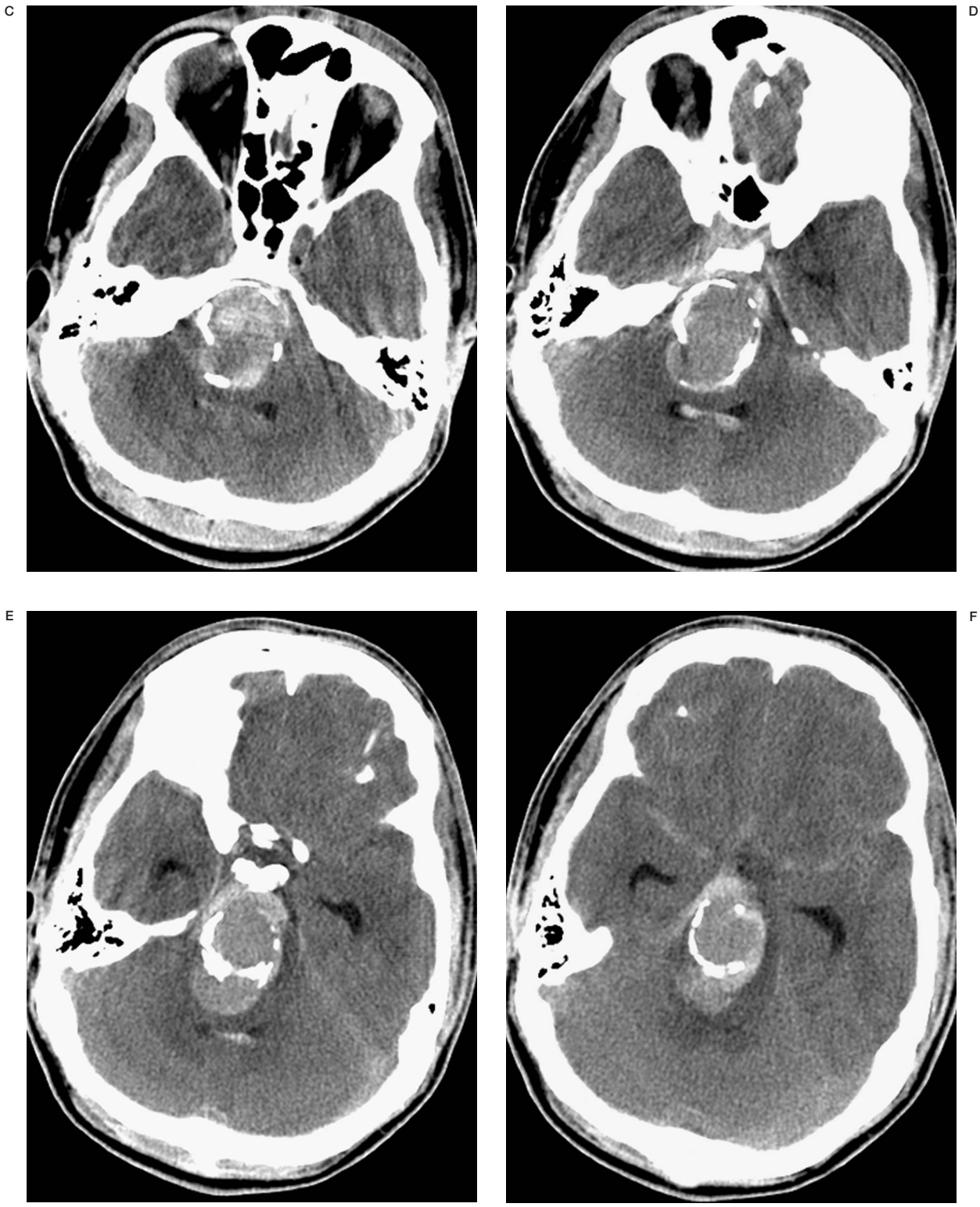


Figure 1 Noncontrast Head CT: Large, irregular, posterior fossa lesion with rim calcification, and partial thrombosis (A-F). Evidence of subarachnoid hemorrhage is seen within the basal cisterns with early ventriculomegaly (E,F). A small amount of intraventricular hemorrhage is seen within the fourth ventricle (C-E).

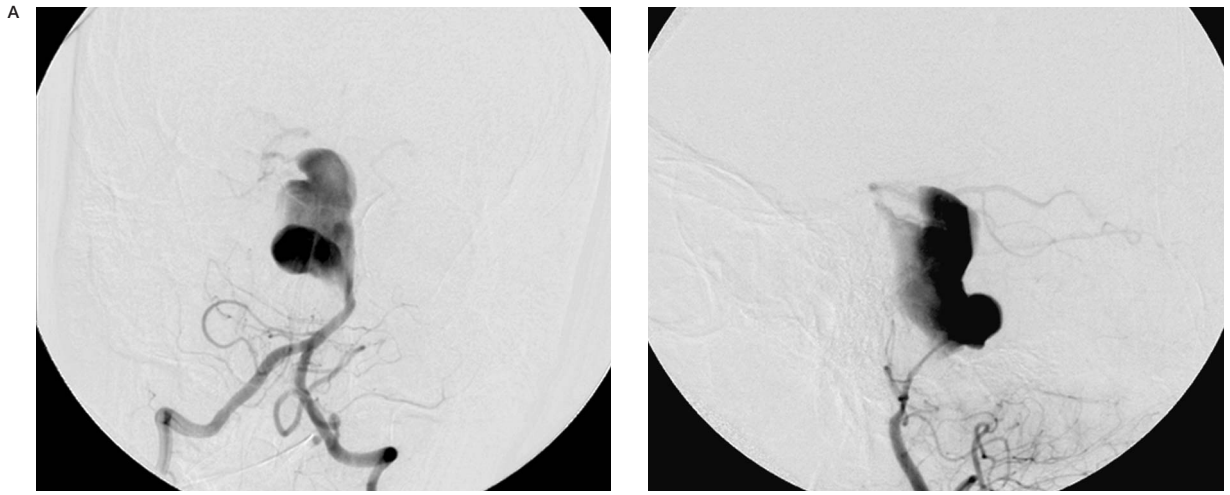


Figure 2 Digitally-subtracted angiogram. Left vertebral injection (A) PA and (B) lateral projections demonstrating a giant, multilobulated distal basilar artery aneurysm.

honor student. Follow-up angiogram showed persistent occlusion of the aneurysm from the circulation (figure 5).

Discussion

Giant fusiform aneurysms of the posterior circulation are associated with a poor prognosis if left untreated [8,5]. The natural history is that of progressive morbidity and mortality. Histopathology studies of these aneurysms have shown attenuation of the smooth muscle layer of the arterial wall with fragmentation of the internal elastic lamina, similar to the destruction of the internal elastic lamina in atherosclerosis [5]. Acute and chronic inflammatory changes in the walls of some aneurysms have been documented [5]. Conditions which weaken the vessel wall such as collagen vascular disease, fibromuscular dysplasia, or maltase deficiency are thought to be contributory factors. Radiological studies of the natural history of incidentally discovered fusiform vertebrobasilar aneurysms have shown that hemorrhage into the vessel is associated with enlargement [5].

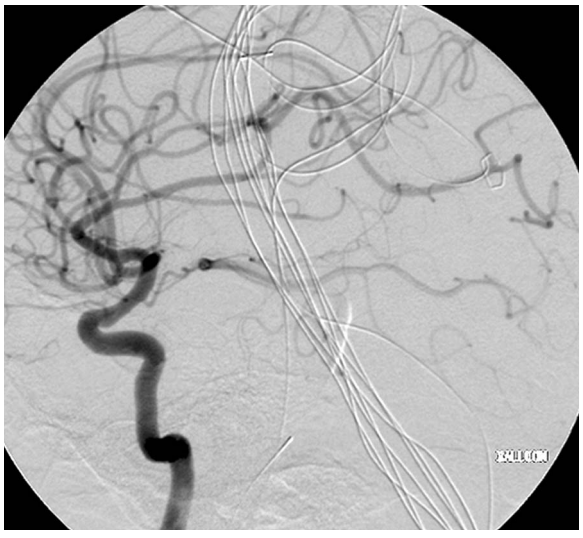


Figure 3 Digitally-subtracted angiogram (Right internal carotid artery injection, lateral) with bilateral vertebral temporary balloon occlusion demonstrating adequate supply through the posterior communicating artery.

Surgical strategies in the treatment of vertebrobasilar aneurysms include wrapping of the aneurysmal wall with various materials to reinforce it, vertebral artery occlusion, basilar artery occlusion, aneurysmal resection with basilar artery reconstruction and trapping of the aneurysm (occlusion of the parent vessel proximal and distal to the aneurysm). Successful endovascular occlusion with coils and balloons has been used alone or in combination with surgical approaches. Endovascular approaches have the advantage of it being easier to carry out test occlusions of the vertebral arteries in non-anesthetized patients. The two most commonly used endovascular treatment strategies are direct coiling of the aneurysm with preservation of parent vessel flow and parent vessel

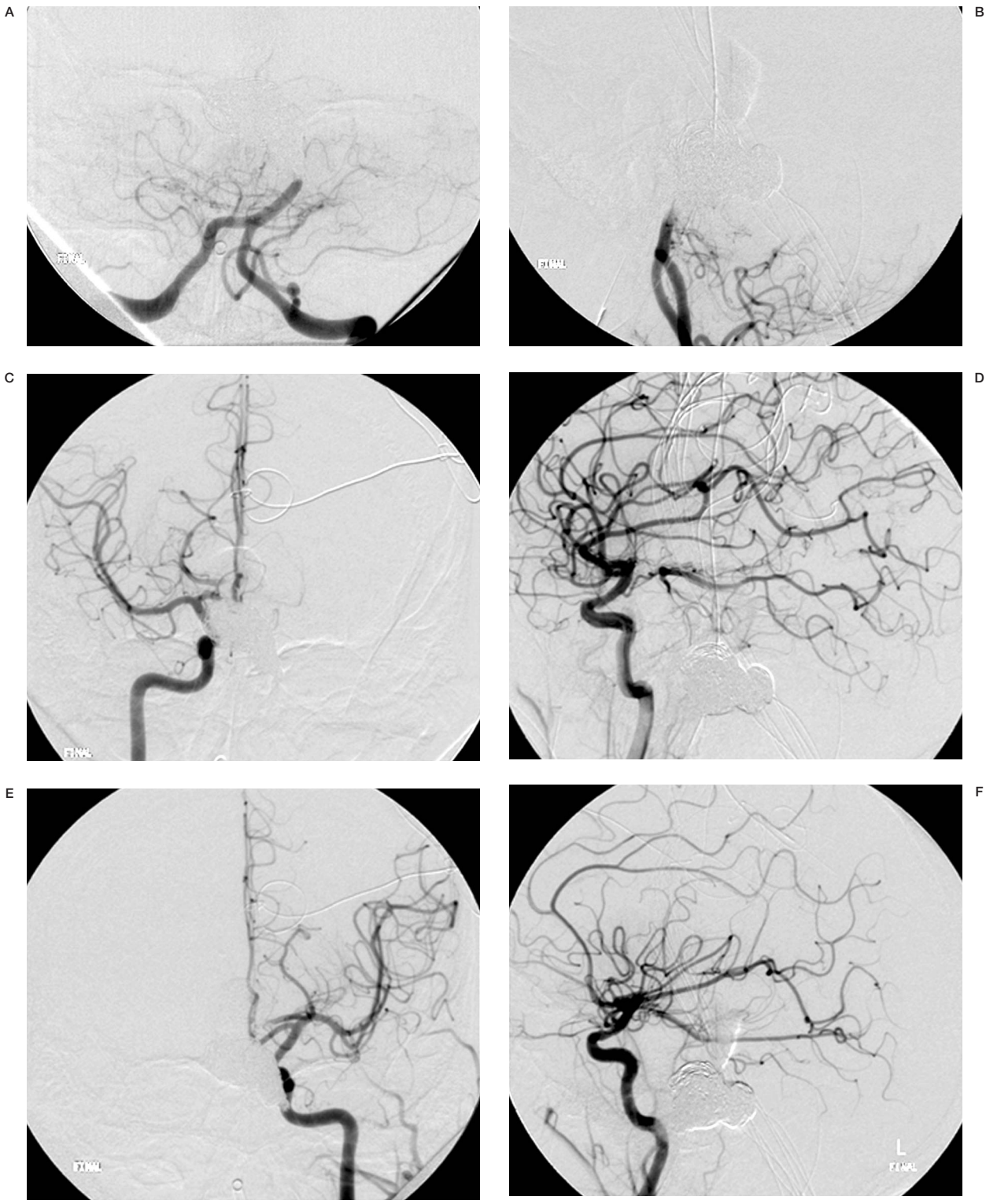


Figure 4 Immediate post-op angiography. (A,B) Left vertebral injection in PA and lateral, respectively, demonstrates complete embolization of the mid-basilar aneurysm with preservation of the proximal basilar artery. (C,D) Right ICA injection in PA and lateral, respectively, shows exclusion of the aneurysm from the circulation with preserved supply to the right posterior cerebral artery and basilar terminus. (E,F) Left ICA injection in PA and lateral, respectively, demonstrates preserved supply to the left posterior cerebral artery and basilar terminus.

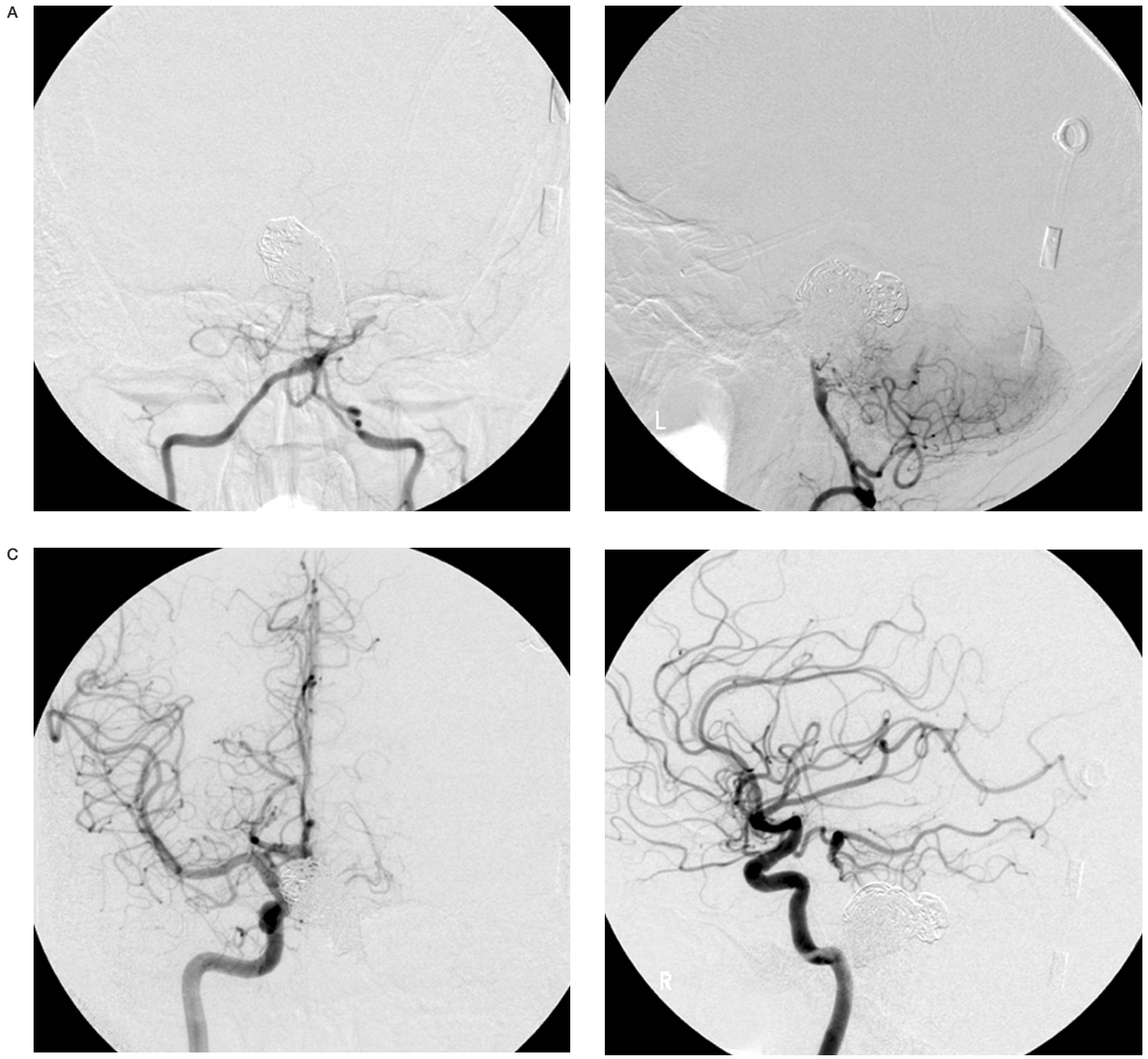


Figure 5 Follow-up angiography at 12 months. (A,B) Left vertebral injection in PA and lateral, respectively, demonstrates persistent exclusion of the aneurysm from the circulation and continued filling of the proximal basilar segment. (C,D) Right ICA injection in PA and lateral, respectively, shows persistent filling of the distal basilar artery and posterior cerebral circulation.

occlusion (PVO). Rarely the two strategies are used together. The former results in better long-term outcome but may be difficult in fusiform aneurysms due to the lack of a definite neck. With complete obliteration of an aneurysm (when obtainable through coil induced thrombosis), the resulting local circulation is more physiological than in parent vessel occlusion.

The most commonly used mechanism of PVO in the posterior circulation is the Hunterian occlusion of one (dominant) or both vertebral arteries. This has been safely carried out in several cases [1,3]. PVO decreases the inflow of blood to the aneurysm.

This prevents further aneurysmal enlargement, promotes aneurysmal thrombosis (from sluggish inflow), and in some studies has caused involution of the aneurysm over a period of time [1,3,5]. Most studies using PVO of one or both vertebral arteries for basilar artery aneurysms have been carried out in awake patients. In these studies, test occlusion of one or both vertebral arteries, was carried out en-

dovascularly for ten to 20 minutes in awake patients, with the guidance of serial neurological examinations. This indeed is an excellent strategy to assess clinically significant collateral inflow from the anterior circulation.

However in comatose patients this strategy fails for obvious reasons. In such circumstances electrophysiological assessment of brainstem function using BAER, SSEP, and MEP can be used as surrogate markers of adequate blood flow to the brain stem, as in our patient. Radiological assessment of adequate collateralization can be done using Alcock's test, which measures the inflow of blood from the vertebrobasilar system to the anterior circulation through the posterior communicating arteries when the internal carotid arteries are externally compressed [1,4]. Tolerance of PVO depends on collateral flow to vital structures. The posterior communicating artery size is an important determinant of the success of PVO.

Steinberg et Al. found that a posterior communicating artery diameter >1 mm significantly improved outcomes in basilar artery and vertebral artery occlusions [6].

In our patient we used the former endovascular strategy. Because of the exceptionally large size of the aneurysm, the serious presentation, and the young age of our patient we opted to directly obliterate the aneurysm, while preserving maximum possible physiologic circulation. Post treatment six month and 12 month follow-up angiograms showed complete obliteration of the aneurysm with no residual blood flow in the mid basilar artery. It is important to note that perforating brain stem branches of the basilar artery are thought to be end arteries and any acute occlusion of these arteries should normally have disastrous consequences. However, due to the fact that our patient remained symptom-free and highly functional in the face of no demonstrable mid basilar artery filling, we hypothesize one of the following two mechanisms:

(a) the presence of the giant basilar aneurysm with circumferential thrombosis may have caused chronic occlusion of his brain stem perforators months to years before he presented to us, and this may have stimulated collateral blood flow to the vital areas of the brain stem.

(b) The aneurysm was very irregular and involved the mid basilar artery primarily. It is possible that all critical brain stem perforators may have arisen from the proximal basilar artery. However, the fact that this patient tolerated temporary bilateral vertebral artery occlusion supports the first hypothesis. While our patient remained completely symptom free at 12 month follow-up, other cases have been documented where residual deficits or poor outcomes have occurred following basilar artery or vertebral artery occlusions [2,3,4].

Conclusions

Endovascular distal basilar artery occlusion can be used as an effective treatment of giant fusiform basilar trunk aneurysms and the safety of this approach can be significantly enhanced with BAER, SSEP, and MEP monitoring in patients with decreased levels of consciousness.

References

1 Boardman P, Byrne JV: Case report: Giant fusiform basilar artery aneurysm: endovascular treatment by flow reversal in the basilar artery. The Br J of Radiology 71: 332-335, 1998.
2 Boet R, Poon WS et Al: Endovascular GDC-Mediated flow reversal for complex posterior circulation saccular aneurysms. Minim Invas Neurosurg 46: 220-227, 2003.
3 Leibowitz R, Do HM et Al: Parent vessel occlusion for vertebrobasilar fusiform and dissecting aneurysms. Am J Neuroradiol 24: 905-907, 2003.
4 O'Shaunessy BA, Getch CC et Al: Progressive growth of a dolichoectatic vertebrobasilar artery aneurysm after complete Hunterian occlusion of the posterior circulation: Case report. Neurosurg 55: 1228-1235, 2004.
5 Omahen D, Findlay JM: A giant fusiform basilar aneurysm treated by bilateral vertebral artery occlusion. J of Clin Neurosci 11(3): 324-328, 2004.
6 Steinberg GK, Drake CG, Peerless SJ: Deliberate basilar or vertebral artery occlusion in the treatment of intracranial aneurysms. Immediate results and long term outcome in 201 patients, J Neurosurg 79: 161-173, 1993.
7 Van Rooij WJ, Sluzewski M et Al: Coiling of saccular basilar trunk aneurysms. Neuroradiology 45: 19-21, 2003.
8 Wenderoth JD, Khangure MS et Al: Basilar trunk occlusion during endovascular treatment of giant and fusiform aneurysms of the basilar artery. Am J Neuroradiol 24: 1226-1229, 2003.

Marlon S. Mathews, M.D.
101 The City Drive South
Bldg 56, Suite # 400, Rt 81
Orange, CA 92868-3298.
E-mail: mmathews@uci.edu

EDITORIAL COMMENT

The authors elegantly show that neurophysiological monitoring (NPM) can be used as an adjunct to angiographic and anatomical analysis in patients who cannot be neurologically evaluated during test occlusion. The concept of using NPM for brainstem function is not new even in the context of aneurysm treatment. Previous papers have discussed the specificity and sensitivity of these methods as treatment monitoring tools and for predicting outcome during open surgical treatment of aneurysms (Ikeda K et Al, 1992, Surg Neurol, *38:445-53; Lopez JR et Al, 1999,* J Neurol Neurosurg, Psychiatry, *66:189-96; Manninen PH et Al, 1994,* Can J Anaesth, *41:92-7; Schick U et Al, 2003,* Acta Neurol Scand, *108:161-69; Schramm J et Al, 1994,* Neurol Res, *16:20-2). A drawback of the methods is certainly that each NPM method measures changes related to a specific anatomical region. It seems, however, that during open surgery, the combination of at least two NPM monitoring methods increases the sensitivity. During endovascular aneurysm treatment, changes in NPM parameters were detected in 26% and altered the treatment strategy in 14% of the cases (Liu AY et Al, 2003,* Am J Neuroradiol, *24:1520-27), which is similar to the experiences during open surgery. The present case adds to the relatively limited experience on NPM in endovascular procedures.*

This case is interesting also from an anatomical perspective since the lesion is located close to the trigeminal point of the basilar artery. The neurological outcome can frequently be predicted by analyzing the lesion, the regional circulation and the collateral circulation induced by temporary vessel occlusion. During development, the trigeminal-basilar artery junction represents the functional termination of caudal division of the internal carotid artery. (Lasjaunias PL et Al, 2001, Surgical Neuroangiography, *Vol.1; Moffat DB, 1961,* J Anat, *95:485-96).*

The upper third of the basilar artery with its perforators therefore probably has an intrinsic preparedness for flow reversal in case of obstruction at the trigeminal point. A parent vessel occlusion there, as described in the present case, will have a relatively low risk of producing neurological symptoms since the embryological circulation pattern, with supply from the anterior circulation to the upper third of the basilar artery and the superior cerebellar arteries, will be restored. The origin of the anterior inferior cerebellar arteries proximal to the occlusion point is also favourable since they will create a sump effect facilitating sufficient flow also to the small perforators originating from the middle segment of the basilar artery. Additional monitoring methods like NPM will, however, further increase the treatment safety in complicated cases like the one presented.

Staffan Holmin MD, PhD
Department of Neuroradiology
Karolinska Hospital
Stockholm, Sweden